Current Comment

S. G. HERSHEY, M.D., *Editor*

Osmolality of Spinal Anesthetic Agents

VINCENT J. SAWINSKI, PH.D.,* ALLEN FRED GOLDBERG, D.D.S.†
NORMA BOONE GOLDBERG, M.D.†

The degree of tissue damage caused by injected regional anesthetic agents as reflected by tissue edema has been demonstrated to be significantly related to the extent of variation of the injected solution from isotonicity.[1] Clinical reports of adverse neurological changes following spinal anesthesia fail to consider this important aspect of the post-injection sequellae.[2] Degeneration and fibrosis of nerve tissue and supporting structure and the accompanying clinical findings of persistent paralysis and anesthesia may not be solely a function of the specific drug administered or the route of administration.

Osmotic pressures of the various commonly used commercially prepared regional anesthetics were measured at body temperature (37° C.) by means of a vapor pressure osmometer (Mechrolab Model 301). Emphasis was especially directed toward the investigation of those drugs administered in proximity to the spinal cord.

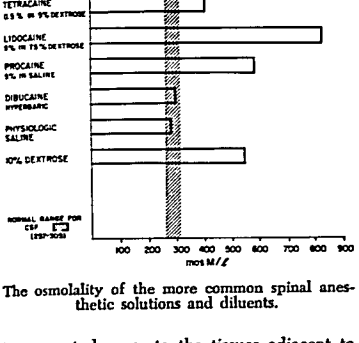
The accompanying table lists the osmolality of many of the commercially available local anesthetic agents and diluents commonly used in administering spinal, caudal, and epidural anesthetics in the form normally injected. The information is also shown graphically in the figure for ease of comparison. It is interesting to note that the majority of the injectable spinal anesthetic solutions are hypertonic with the mean value for the osmolality of replicate samples of most solutions falling outside the normal range for the osmolality of cerebrospinal fluid, that is, 257 to 305 milliosmoles per liter.[3] It should also be remembered that while the osmolality of some spinal anesthetic solutions does indeed fall within the normal range for cerebrospinal fluid, this does not preclude tissue damage which is secondary due to the toxicity of the injected solution.

From the data cited, it would seem reasonable to attempt to use regional anesthetic solutions which have osmotic pressure within or at least close to the normal range in an effort

OSMOLALITY OF SPINAL ANESTHETIC AGENTS

Anesthetic Solution	Mean Osmolality (Milliosmoles/liter)
1. 0.0025% dibucaine in 10% dextrose	299
2. 2.5% hexylcaine in 10% dextrose	792
3. 1% mepivacaine (caudal and epidural)	300
4. 2% mepivacaine (for caudal and epidural)	263
5. mepivacaine, isobaric, for spinal anesthesia	290
6. 1% procaine	276
7. 2% procaine	310
8. 5% procaine in saline	565
9. 1% lidocaine	294
10. 2% lidocaine	321
11. 1.5% lidocaine with 1/100,000 epinephrine	320
12. 5% lidocaine in 7.5% dextrose	825
13. 3% citanest in 0.6% sodium chloride	457
14. 1% tetracaine	303
15. 0.5% tetracaine in 5% dextrose	406

* Veterans Administration Hospital, Hines, Illinois, and Loyola University, Chicago, Illinois.
† Lutheran General Hospital, Park Ridge, Illinois, and University of Illinois, College of Medicine, Chicago, Illinois.



The osmolality of the more common spinal anesthetic solutions and diluents.

to prevent damage to the tissues adjacent to the initial site of anesthetic solution deposition. While this tissue damage is usually reversible, it should be remembered that this is not always true.

Summary

An extensive survey of the osmotic pressures of commonly used commercial spinal anesthetic solutions was accomplished. It was found that most of the drug solutions studied had osmolalities which exceeded the usually accepted physiologic range of 257 to 305 milliosmoles per liter for cerebrospinal fluid.

References

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Brachial Plexus Infiltration with Dilute Phenol Solution in the Management of Upper Extremity Spasticity

Jordan Katz, M.D.,* Leslie K. Knott, M.D.,† Daniel J. Feldman, M.D.‡

Injection of the brachial plexus with phenol has not been reported as an adjunct in the management of patients with spastic arms. In the course of our investigations of phenol blocks on peripheral nerves in spastic extremities,** we decided to attempt brachial plexus infiltration with 3 per cent phenol in water solutions.

Three patients were studied. Brachial plexus block was done to attempt to alleviate disabling spasticity and clonus and to increase muscle function. The supraclavicular approach was used twice and the axillary technique once. One patient had excellent relief and increased function, the second showed increased function but no objective change in spasticity, and the third no changes at all. The first patient only will be presented here:

A 29 year old man was referred to us for evaluation and treatment of a spastic left arm. Two years previously the patient had developed subacute bacterial endocarditis with embolism to the right hemisphere and a resulting left sided hemiparesis. Functional return was gradual and at the time we first saw the patient he had a poor to fair return of motor power. His main problem was the persistences of spasticity and clonus in the left arm. Using a 0 to 4 plus scale, with 4 plus being intense spasticity, our initial evaluation of the patient was as follows: There was 2–3 plus spasticity in the abductors, abductors and rotators of the shoulder; 2–3 plus spasticity in the flexors and extensors of the elbow and wrist; the metacarpalphalangeal joints had 2 plus spasticity and the interphalangeal joints were 3–4 plus.

* Department of Anesthesia, Stanford University School of Medicine.
† Division of Rehabilitation, Stanford University School of Medicine.
‡ Division of Rehabilitation, Stanford University School of Medicine, Stanford, California.
** Katz, J., Knott, L. W., and Feldman, D. J.: Peripheral nerve injections with phenol in the management of spastic patients, submitted to Arch. Phys. Med.